Atrial and ventricular function after cardioversion of atrial fibrillation

Changsheng Xiong, Christine Sonnhag, Eva Nylander, Bengt Wranne

Abstract

Objective—Previous studies on atrial recovery after cardioversion of atrial fibrillation have not taken into account new knowledge about the pathophysiology of transmitral and tricuspid flow velocity patterns. It is possible to shed further light on this problem if atrioventricular inflow velocity, venous filling pattern, and atrioventricular annulus motion are recorded and interpreted together.

Design—Prospective examinations of mitral and tricuspid transvalvar flow velocities, superior caval and pulmonary venous filling, and mitral and tricuspid annulus motion were recorded using Doppler echocardiography. Examinations were performed before and 24 hours, 1 month, and 20 months after cardioversion.

Setting—Tertiary referral centre for cardiac disease with facilities for invasive and non-invasive investigation.

Patients—16 patients undergoing cardioversion of atrial fibrillation in whom sinus rhythm had persisted for 24 hours or more.

Results—Before conversion there was no identifiable A wave in transvalvar flow recordings. The total motion of the tricuspid and mitral annulus was subnormal and there was no identifiable atrial component. Venous flow patterns in general showed a low systolic velocity. After conversion, A waves and atrial components were seen in all patients and increased significantly ($P < 0.01$) with time. There was a similar time course for the amplitude of annulus atrial components, an increased systolic component of venous inflow, an increased A wave velocity, and a decreased E/A ratio of the transvalvar velocity curves. The ventricular component of annulus motion was unchanged. Changes in general occurred earlier on the right side than the left.

Conclusions—This study indicates that, in addition to the previously known electromechanical dissociation of atrial recovery that exists after cardioversion of atrial fibrillation, there may also be a transient deterioration of ventricular function modulating the transvalvar inflow velocity recordings. Function on the right side generally becomes normal earlier than on the left. Integration of information from transvalvar inflow

curves, annulus motion, and venous filling patterns gives additional insight into cardiac function.

(Br Heart J 1995;74:254-260)

Keywords: cardioversion; atrial fibrillation; cardiac function

Effective atrial mechanical contraction may be absent or weak for days or weeks after restoration of sinus rhythm by cardioversion of atrial fibrillation. This was noted with invasive techniques analysing pressure tracings from the right and left atria¹ and with non-invasive techniques such as apexcardiography and recording of jugular venous pulses.² More recent studies using M mode and cross sectional echocardiographic techniques³⁻⁵ and the pulsed wave Doppler technique have concentrated either on the left heart⁶⁻⁸ or the right heart.⁹

The results from comparisons of time course of recovery of left and right atrial function are conflicting, some indicating that right atrial function generally recovers faster than left atrial function,^{1,2} while another study showed no difference.¹⁰ A recent study indicates that time of recovery for left atrial function is related to duration of atrial fibrillation before cardioversion.¹¹

At the time of the above Doppler-echocardiographic studies the pathophysiological background of changes in the mitral inflow curve was not fully understood; an increased A wave velocity and a decreased E/A ratio of the mitral inflow curve were interpreted as signs of increased atrial activity. We now know that factors determining the mitral inflow curve are more complex and reflect ventricular as well as atrial properties, in addition to effects of impedance of the mitral ostium.¹²⁻¹⁴ A decreasing E/A ratio may be interpreted as a sign of improved atrial function but also of a relaxation abnormality of the ventricle. After cardioversion of atrial fibrillation, diastolic ventricular function may hypothetically be impaired with increased filling pressures. This would give a pseudonormal mitral inflow curve. With time, the filling pressure may become normal. If a relaxation abnormality of the ventricle then exists, the pseudonormal pattern would change to a pattern of relaxation abnormality—that is, the A wave velocity would increase and the E/A ratio decrease. The earlier reported pattern⁶⁻⁹ may thus be explained by changes in ventricular properties in addition to or instead of

Linköping Heart Centre, Department of Clinical Physiology, University Hospital, Linköping, Sweden

C Xiong
E Nylander
B Wranne

Linköping Heart Centre, Department of Cardiology, University Hospital, Linköping
C Sonnhag

Correspondence to:
Dr B Wranne, Department of Clinical Physiology, University Hospital, S-581 85 Linköping, Sweden.

Accepted for publication
30 January 1995

changes in atrial properties. The different time course between the right and left heart can hypothetically be explained in this way also.

Further light can be shed on this problem if atrioventricular inflow velocity, venous filling patterns, and atrioventricular annulus motion are recorded and interpreted together. To our knowledge, this approach has not been applied earlier in this context. The aim of our study was therefore to establish whether there is a difference between the right and left heart when Doppler-echocardiographic methods are used and also to distinguish the atrial and ventricular contribution to these changes.

Patients and methods

PATIENTS

We included 16 patients under 70 (11 men, five women; mean age 63 years, range 47 to 68) who were undergoing electric cardioversion for atrial fibrillation and who maintained sinus rhythm for at least 24 hours. The underlying cardiovascular or systemic disorders predisposing to atrial fibrillation were ischaemic heart disease in two patients, thyrotoxicosis in two, hypertension in two, alcoholism in one, and lone atrial fibrillation in nine. None of the patients had more than mild mitral regurgitation. Three patients were classed as being in New York Heart Association functional class I; nine were in class II and four in class III. The mean duration of atrial fibrillation was 5 months (2 months to 1 year). Onset was defined as onset of symptoms prompting admission, or in symptom free patients the first electrocardiogram showing atrial fibrillation. Before cardioversion all patients took warfarin for at least four weeks. Three patients were taking diuretics, 10 digitalis, seven β blockers or sotalol, and four verapamil. Three patients were taking no drugs. Cardioversion was performed under general anaesthesia with intravenous propofol while the patient was breathing spontaneously. Treatment during the observation period after cardioversion was essentially the same as before the procedure. Patients who had significant valvar disease, a valve prosthesis, implanted pacemakers, or severe ventricular dysfunction were excluded. All subjects gave informed consent to the protocol, which was approved by the local committee for the protection of human subjects.

ECHOCARDIOGRAPHIC STUDY

Echocardiography was performed before and 24 hours after cardioversion and after 1 month and 20 months (range 6–26) if patients maintained sinus rhythm.

Standard M mode, cross sectional, and pulsed Doppler echocardiograms were obtained in the partial left lateral supine position using an Acuson ultrasound imaging system (128 XP-10, Acuson Corporation, California) equipped with a 2.5 MHz phased array transducer; a simultaneous electrocardiogram and respiratory trace on videotape and strip chart at paper speeds of 50 or

100 mm/s were recorded for subsequent analysis of data.

Pulsed wave Doppler transmitral and transtricuspid flow velocities were recorded from the apical four chamber view with the sample volume positioned between the tips of the atrioventricular valve leaflets. Aortic subvalvar velocities were recorded from the apical five chamber view. Flow in the superior caval vein was examined from a supraclavicular window with the sample volume at a depth of 6 to 7 cm. The four chamber view that best showed the left atrium and pulmonary veins was used to record pulmonary vein velocities, the sample volume being at the entrance of a right pulmonary vein into the left atrium. Angle correction was not performed. A low filter setting was used to include low velocity signals. Tricuspid and mitral annulus motion were recorded at their lateral and septal points by cross sectional directed M mode echocardiography from the apical four chamber view.¹⁵ All data were recorded during quiet respiration and during episodes of expiratory apnoea lasting 8–10 s. A zoom technique was used to expand the selected region.

MEASUREMENT AND ANALYSIS

The readings were performed visually by means of a Panasonic video cassette recorder (Model AG-7330) with playback and slow motion capabilities. Tracings were manually traced with the cardiac calculation software of the Acuson imaging system. For the recordings before cardioversion, heart rate was counted from the tapes or strip charts. The second beat from two consecutive beats whose RR interval corresponded to the mean RR interval was chosen for analysis of cavity dimensions and Doppler velocities. Heart rate was not corrected. All the findings reported are the average of three to five beats. Technically inadequate recordings were excluded.

Cavity dimensions were measured from standard M mode studies and the cross sectional four chamber view. Ventricular long and short axes, ventricular inflow tract, and atrial long and short axes views were selected and measured from end diastolic and end systolic frames. Aortic subvalvar area was calculated from the subvalvar diameter, assuming that a circular shape and volume flow was obtained as the product of area and subvalvar systolic velocity integral. Tricuspid and mitral valve diastolic flow velocities were analysed. E and A wave peak velocities were measured and their ratio calculated. Total excursion and the atrial component of tricuspid lateral and septal and mitral annular lateral points motion were measured by the leading edge technique.¹⁵ The atrial component contribution to total annulus motion was calculated. Systolic and diastolic forward flow velocities and time velocity integrals of the superior vena cava and a pulmonary vein were measured. Systolic to diastolic flow ratio was calculated. Peak reverse flow A wave and V wave in superior vena cava were also determined when present.

STATISTICAL ANALYSIS

Results are presented as means (SD) and as ranges. Analysis of variance was used when data from the four periods of observation were compared. If a significant change was found, Student's paired *t* test was used for statistical analysis of data. The unpaired *t* test was used for comparisons between normal subjects and patients. *P* values obtained in *t* tests refer to two tailed tests, and *P* < 0.05 was considered significant.

Results

Six of the 16 patients who had sinus rhythm 24 hours after cardioversion later reverted to atrial fibrillation, five patients during the first month and one patient during the second. Ten patients still maintained sinus rhythm at the 20 month examination. Cardioversion resulted in a significant reduction in heart rate. An increased stroke volume was noted at the 20 month examination (fig 1). There were no changes in cross sectional or M mode dimensions of the cavity during the period after conversion, with the exception of a small but significant (*P* < 0.01) decrease in the anterior-posterior diameter of the left atrium

Figure 1 Heart rate and stroke volume before and 24 hours, 1 month, and 20 months after cardioversion. Bars indicate one SD; numbers in parentheses are number of patients; **P* < 0.05, ***P* < 0.01.

Some echocardiographic data before and after cardioversion. Values are means (SD)

	Before	After			
		24 hours	1 month	20 months	
MV(A) (m/s)	—	0.26 (0.11)	0.53 (0.15)‡	0.56 (0.20)‡	
TV(A) (m/s)	—	0.17 (0.08)	0.26 (0.10)‡	0.27 (0.08)§	
Man(a) (mm)	—	2.8 (1.0)	6.5 (1.8)‡	5.8 (1.0)‡	
Tan(a) (mm)	—	7.6 (1.9)	10.0 (2.4)§	9.7 (2.5)§	
Pulmonary vein:					
SV (m/s)	0.17 (0.06)	0.39 (0.15)*	0.59 (0.16)*‡	0.54 (0.18)*§	
SVTI (m)	0.03 (0.01)	0.09 (0.04)*	0.17 (0.06)*‡	0.14 (0.05)*‡	
DV (m/s)	0.53 (0.11)	0.60 (0.15)	0.45 (0.15)§	0.43 (0.10)†	
DVTI (m)	0.12 (0.02)	0.19 (0.19)	0.11 (0.04)	0.11 (0.03)	
Systolic fraction	0.18 (0.04)	0.35 (0.12)*	0.60 (0.10)*‡	0.55 (0.11)*‡	
Superior vena cava:					
SV (m/s)	0.29 (0.18)	0.38 (0.14)†	0.39 (0.12)	0.36 (0.11)	
SVTI (m)	0.06 (0.02)	0.11 (0.04)*	0.12 (0.03)*§	0.12 (0.03)*	
DV (m/s)	0.31 (0.16)	0.24 (0.09)	0.22 (0.06)	0.23 (0.08)	
DVTI (m)	0.07 (0.03)	0.06 (0.02)	0.06 (0.02)	0.06 (0.02)	

**P* < 0.01 compared with before; †*P* < 0.05 compared with before; ‡*P* < 0.01 compared with 24 hours, §*P* < 0.05 compared with 24 hours.

DV, diastolic velocity; DVTI, diastolic velocity time integrals, Man(a), mitral annulus motion atrial component; MV(A), transmitral A wave velocity; SV, systolic velocity; SVTI, systolic velocity time integral; Tan(a), tricuspid annulus motion atrial component; TV, transtricuspid A wave velocity.

from 41 (5) mm 24 hours after the procedure to 37 (4) mm one month afterwards.

Before conversion there was no identifiable A wave in the transtricuspid and transmitral flow recordings. Twenty four hours after conversion, atrial filling waves were seen in all patients and increased significantly from this occasion until the one month and late controls (Table). The transmitral E/A velocity ratio was above 1 at the 24 hour control and decreased significantly until both the one month and late controls (fig 2). Before conversion the total motion of the tricuspid and mitral annulus were significantly lower than that seen in normal subjects of similar age (*P* < 0.001) and there was no identifiable atrial contribution. After conversion there was a significant increase in motion of both the tricuspid and mitral annulus. In both annuli

Figure 2 E/A ratio of mitral and tricuspid transvalvar velocities 24 hours, 1 month, and 20 months after cardioversion. Bars indicate one SD; numbers in parentheses are numbers of patients; **P* < 0.05, ***P* < 0.01.

Figure 3 Total motion and ventricular and atrial components of annulus motion before and 24 hours, 1 month, and 20 months after cardioversion. Bars indicate one SD; numbers in parentheses are numbers of patients; **P* < 0.05, ***P* < 0.01.

Figure 4 Typical recordings of tricuspid annulus motion before (top) and 24 hours (middle) and 1 month (bottom) after cardioversion. Before cardioversion there was no identifiable atrial component. Distance between open arrows is the atrial component; distance between closed arrows is total motion. The difference between total motion and atrial component gives the ventricular component.

Figure 5 Transvalvar A wave velocity in relation to atrial contribution of valve motion 24 hours (○), 1 month (△), and 20 months (●) after cardioversion.

Figure 6 Typical recording of pulmonary venous flow before and after cardioversion. D, diastolic flow; S, systolic flow.

this increase was due to the contribution of atrial activity, the motion related to ventricular relaxation being unchanged (9 (2) mm before, 10 (2) at 24 hours, 8 (2) at one month, and 10 (2) at 20 months for the ventricular component of the mitral valve and 16 (4), 14 (4), 14 (3), and 14 (3) mm for the tricuspid annulus). The changes were similar for the lateral and septal aspects, so data from the lateral points alone are reported. There was a different time course between the two sides, the atrial component of the tricuspid annulus increasing earlier than that of the atrial component of the mitral annulus (fig 3). Typical recordings of tricuspid annulus motion are given in fig 4. The relation between changes of annulus motion atrial contribution and transvalvar A wave velocity is shown in fig 5.

Pulmonary venous flow patterns during atrial fibrillation showed a low systolic velocity, the systolic to diastolic ratio always being below 1 (fig 6). The systolic contribution, expressed as systolic fraction (systolic time integral/(systolic + diastolic time integral)) was 0.18 (0.04) (table). The systolic contribution increased with time after cardioversion, but 24 hours later it was still < 0.4 in six of the patients (table). During atrial fibrillation it was often difficult to separate systolic from diastolic flow in the superior vena cava because of overlapping (fig 7, A). In most patients the diastolic part dominated, while in four patients the systolic part dominated (fig 7, B). Twenty four hours after cardioversion the systolic velocity and the systolic

Figure 7 Two typical recordings of flow patterns from the superior vena cava showing that systolic flow velocity sometimes is higher than diastolic before cardioversion. A: Biphasic flow with diastolic predominance. B: Systolic predominance, which never occurred in recordings from the pulmonary veins during atrial fibrillation. D, diastolic flow; S, systolic flow.

to diastolic velocity ratio of the flow in the superior vena cava had increased significantly and did not increase further (fig 8). Thus there was a different time course for venous return to the right and left heart, the changes on the right side showing earlier.

There were no appreciable flow reversals at the end of diastole before conversion. All recordings in pulmonary vein and superior vena cava showed identifiable reverses in the atrial component 24 hours after cardioversion.

Discussion

Our data contribute to the understanding of haemodynamic adaptation after conversion of atrial fibrillation to sinus rhythm by combining the information contained in venous inflow patterns, transvalvar flow velocities, and annulus motion and by recording the dif-

Figure 8 Ratios of venous systolic to diastolic velocities and velocity time integrals before and 1 month and 20 months after cardioversion. Bars indicate one SD; numbers in parentheses are numbers of patients; * $P < 0.05$, ** $P < 0.01$.

ferent time course of recovery between the left and right heart.

ATRIAL RECOVERY

Published evidence favours the concept that there is an electromechanical dissociation of atrial recovery after successful cardioversion of atrial fibrillation.¹⁻¹¹ This conclusion has to a great extent been drawn from Doppler studies in which increased A wave velocities of the mitral or tricuspid inflow curves were interpreted as signs of improved atrial function. We now know that the pathophysiology of atrioventricular inflow is more complex, being influenced by both atrial and ventricular properties in addition to the influence of the impedance of the ostium. A change from an apparently normal curve to one of delayed ventricular relaxation with decreasing E/A ratio may in previously normal subjects be a sign of deterioration, while for patients who have had a myocardial infarction it may represent an improved status with decreasing preload. A third interpretation would be that of improved atrial function. Interpretation of the atrioventricular inflow pattern is facilitated if it is combined with analysis of venous inflow

and annular motion,¹⁴⁻¹⁶ as we have done in this study.

Annular motion and its relation to venous flow has been studied in normal subjects and patients.^{15,16} Normal values for tricuspid motion and its relation to central venous flow were reported by Hammarström *et al.*¹⁵ A decreased mitral annulus motion is associated with left ventricular dysfunction and accompanied by an altered pulmonary venous inflow pattern.¹⁶ Decreased tricuspid annulus motion and lack of systolic component in the central venous flow was reported after cardiac surgery.¹⁷ Ochi *et al* investigated tricuspid annulus motion and systolic venous return after cardioversion of atrial fibrillation and noticed increases in systolic forward flow in the superior caval vein and the ratio of late diastolic to total excursion of the tricuspid annulus. They concluded that atrial relaxation rather than systolic descent of the tricuspid annulus was the predominant factor determining systolic forward flow in the superior vena cava.

We found an increased atrial contribution of annulus motion with time after cardioversion of atrial fibrillation. The time course differed, with an earlier increase in the atrial contribution of the tricuspid annulus motion than that of the mitral annulus; the ventricular portion was unchanged for both. We have interpreted this as a sign of atrial recovery. We found a similar time course to that of annular motion for the increase in transvalvar A wave velocity, decrease in E/A ratio, and increase in systolic contribution of venous filling. Atrial recovery is thus likely to be a major component of the change in transvalvar flow pattern with time after cardioversion.

VENTRICULAR FUNCTION

Using radionuclide techniques Kieny *et al* reported increased ejection fractions five months after successful cardioversion in patients with atrial fibrillation and idiopathic dilated cardiomyopathy.¹⁸ Miwa *et al* found increased fractional shortening 2–12 months after cardioversion using M mode echocardiography.¹⁰ Alam *et al* showed that total annulus motion is related to ventricular function expressed as ejection fraction and also that mitral annulus motion amplitude increases after cardioversion, interpreting this as a sign of improved left ventricular function.⁵ The importance of the contribution of annulus motion to cardiac performance has long been known.^{19,20} Lundbäck even claims that this together with the modulating influence of the interventricular septal motion is the most important mechanism of cardiac performance.²¹ Our data show that the increase in amplitude of annulus motion after cardioversion is caused by an increased atrial component. Nevertheless, this causes an extra displacement of the annulus towards the base in diastole, thereby stretching the ventricular muscle fibres, causing them to contract more vigorously according to the Frank Starling principle.

During atrial fibrillation pulmonary venous

systolic fraction was below 0.4 in all patients. One day after cardioversion it was still below 0.4 in almost half of them. It has recently been shown in other patient groups that a ratio below 0.4 is highly indicative of an increased left ventricular filling pressure.²² A diastolic relaxation abnormality of the ventricle in combination with increased left atrial pressure gives a “pseudonormal” (E > A) transmitral flow velocity. The pattern found 24 hours after cardioversion with an E/A ratio > 1 changing to one with E/A ratio < 1 at the later controls may thus be a pseudonormal pattern (increased left atrial pressure) changing to one of a relaxation abnormality (normal left atrial pressure). The time course of E/A ratio after cardioversion may thus be influenced by changes in left ventricular properties in addition to changes in atrial mechanical activity. This is also in line with recent animal studies showing markedly increased left ventricular filling pressures in combination with signs of delayed diastolic relaxation after direct current counter shock.²³

DIFFERENT TIME COURSE FOR CHANGES IN LEFT AND RIGHT HEART

Changes after cardioversion were qualitatively similar for the left and right heart, but the time course was different. Thus, changes on the right side—that is, amplitude of the atrial component of tricuspid annulus motion—and the ratio of systolic venous velocity to diastolic venous velocity all improved earlier than corresponding signs on the left side (figs 3 and 7, table). The results agree with those from studies performed separately on the left^{3,7,8} or right heart,⁹ or on both sides with similar techniques,²⁴ or with invasive¹ or pulse recording techniques,² but they are at variance with recent results.¹⁰ The delay on the left side has been attributed to the causes of diseases such as chronic rheumatic disease that affect the left side,^{1,2} but it also varies with duration of atrial fibrillation before cardioversion.¹¹ Gelder *et al* found that the decrease in atrial size after cardioversion varied.²⁴ Our data do not allow any further conclusions about the cause of the different time course. One point of interest, however, is that during atrial fibrillation pulmonary venous flow never showed a dominating systolic wave, while for the right heart the systolic wave dominated in five of the 16 patients. The tricuspid annulus is normally displaced more than the mitral annulus during systole,¹⁵ and this was also true for our patients during atrial fibrillation. The suction effect of the systolic descent of the tricuspid annulus is thus likely to be larger than that of the mitral annulus, causing a more pronounced systolic emptying of the superior caval vein than of the pulmonary veins.

LIMITATIONS OF STUDY

The number of patients is rather small but the findings are consistent. Inclusion of more patients would not change the answers to the questions addressed here. It would have been a great advantage to have pressure monitoring of the patients during the early period after

cardioversion in order to separate changes in atrial and ventricular properties. This was, however, deemed unethical to do.

CONCLUSION

The present study provides data that improve the understanding of haemodynamic changes after cardioversion of atrial fibrillation. The change in transmitral flow pattern seen after successful cardioversion seems to be mainly caused by atrial electromechanical dissociation, but changes in ventricular filling pressure may modulate the observed patterns.

Financial support was given by Swedish Medical Research Council (grant 9481), the Swedish Heart Lung Foundation, and the Research Foundation of the University Hospital of Linköping. We thank Mrs Gudrun Lövdahl and Mrs Birgit Käll for skilful technical help.

- 1 Rowlands DJ, Logan WFWE, Howitt G. Atrial function after cardioversion. *Am Heart J* 1967;74:149–60.
- 2 Ikram H, Nixon PGF, Arcan T. Left atrial function after electrical conversion to sinus rhythm. *Br Heart J* 1968;30:80–3.
- 3 de Maria AN, Lies JE, King JF, Miller RR, Amsterdam EA, Mason DT. Echographic assessment of atrial transport, mitral movement and ventricular performance following electroversion of supraventricular arrhythmias. *Circulation* 1975;51:273–82.
- 4 Orlando JR, Herick R, Aronow WS, Olson HG. Hemodynamics and echocardiograms before and after cardioversion of atrial fibrillation to normal sinus rhythm. *Chest* 1979;76:521–6.
- 5 Alam M, Thorstrand C. Left ventricular function in patients with atrial fibrillation before and after cardioversion. *Am J Cardiol* 1992;69:694–6.
- 6 Shapiro EP, Effron MB, Lima S, Ouyang P, Siu CO, Bush D. Transient atrial dysfunction after conversion of chronic atrial fibrillation to sinus rhythm. *Am J Cardiol* 1988;62:1202–7.
- 7 Manning WJ, Leeman DE, Gotch PJ, Come PC. Pulsed Doppler evaluation of atrial mechanical function after electrical cardioversion of atrial fibrillation. *J Am Coll Cardiol* 1989;13:617–23.
- 8 Manning WJ, Silverman DI, Katz SE, Douglas PS. Atrial ejection force: a noninvasive assessment of atrial systolic function. *J Am Coll Cardiol* 1993;22:221–5.
- 9 Ochi H, Izumi S, Murakami R, Shimada T, Morioka S, Moriyama K. Superior vena cava flow and tricuspid annular motion after cardioversion of atrial fibrillation, and role of right atrial relaxation on systolic venous return. *Am J Cardiol* 1991;68:1335–9.
- 10 Miwa H, Arakawa M, Kagawa K, et al. Time-course of recovery of atrial contraction after cardioversion of chronic atrial fibrillation. *Heart Vessels* 1993;8:98–106.
- 11 Manning WJ, Silverman DI, Katz SE, Riley MF, Come PC, Doherty RM, et al. Impaired left atrial mechanical function after cardioversion: relation to duration of atrial fibrillation. *J Am Coll Cardiol* 1994;23:1535–40.
- 12 Nishimura RA, Housmans PR, Hatle LK, Tajik AJ. Assessment of diastolic function of the heart: background and current applications of Doppler echocardiography. I. Physiologic and pathophysiologic features. *Mayo Clin Proc* 1989;64:71–81.
- 13 Nishimura RA, Housmans PR, Hatle LK, Tajik AJ. Assessment of diastolic function of the heart: background and current applications of Doppler echocardiography. II. Clinical studies. *Mayo Clin Proc* 1989;64:182–204.
- 14 Appleton CP, Hatle LK. The natural history of left ventricular filling abnormalities: assessment by two-dimensional and Doppler echocardiography. *Echocardiography* 1992;9:437–57.
- 15 Hammarström E, Wranne B, Pinto FJ, Puryear J, Popp RL. Tricuspid annular motion. *J Am Soc Echocardiogr* 1991;4:131–9.
- 16 Keren G, Sonnenblick EH, LeJemtel TH. Mitral annulus motion. Relation to pulmonary venous and transmitral flows in normal subjects and in patients with dilated cardiomyopathy. *Circulation* 1988;78:621–9.
- 17 Wranne B, Pinto FJ, Hammarström E, Goar FG, Puryear J, Popp RL. Abnormal right heart filling after cardiac surgery: time course and mechanisms. *Br Heart J* 1991;66:435–42.
- 18 Kiény JR, Sacrez A, Facello A, et al. Increase in radionuclide left ventricular ejection fraction after cardioversion of chronic atrial fibrillation in idiopathic dilated cardiomyopathy. *Eur Heart J* 1992;13:1290–5.
- 19 Hamilton WF, Rompf JH. Movements of the base of ventricle and relative constancy of cardiac volume. *Am J Physiol* 1932;102:559–65.
- 20 Rushmer RF. *Cardiovascular dynamics*. 3rd ed. London: Saunders, 1970:33–56.
- 21 Lundbäck S. Cardiac pumping and function of the ventricular septum. *Acta Physiol Scand Suppl* 1986;550:19–32.
- 22 Rossvoll O, Hatle LK. Pulmonary venous flow velocities recorded by transthoracic Doppler ultrasound. Relation to left ventricular diastolic pressure. *J Am Coll Cardiol* 1993;21:1687–96.
- 23 Yasuda S, Shishido T, Goto Y. Direct-current counter-shock induces predominant diastolic dysfunction without major changes in contractile efficiency or oxygen cost of contractility [abstract]. *Journal of Cardiovascular Diagnostics and Procedures* 1994;12:120.
- 24 Gelder IC, Crijns HJ, Gilst WH, Hamer HPM, Lie KI. Decrease of right and left atrial sizes after direct-current electrical cardioversion in chronic atrial fibrillation. *Am J Cardiol* 1991;67:93–5.